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GRAM-NEGATIVE SEPTICEMIA IN AMERICAN ALLIGATORS (*ALLIGATOR MISSISSIPPIENSIS*)

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ABSTRACT: Six species of bacteria (family Enterobacteriaceae) not commonly reported as associated with disease in American alligators (*Alligator mississippiensis*) were documented, suggesting that *Aeromonas* is not the only bacterium responsible for septicemia in crocodilians. These included *Citrobacter freundii*, *Enterobacter agglomerans*, *Proteus* sp., *Morganella morganii*, *Serratia marcescens*, and *Klebsiella oxytoca*. Clinical signs of disease included intensive basking, anorexia, lethargy, flaccid limb paralysis, stomatitis, and dermatitis. Our data indicated that early treatment with broad-spectrum antibiotics was preferable to waiting for sensitivity results.

INTRODUCTION

Following the impending removal of American alligators from state and federal endangered species lists (Anonymous, 1985), these animals are likely to become increasingly important in experimental studies. Large numbers of alligators are already raised commercially in Florida and Louisiana. It is therefore necessary for investigators and breeders to be aware of potential bacterial infections and the clinical signs of these infections in order to determine the proper treatment of the animals. However, bacterial diseases in crocodilians have not been well documented (Jacobson, 1984). Among the bacteria associated with disease in crocodilians are *Erysipelothrix insidiosa*, *Pasteurella multocida*, and *Staphylococcus aureus* (Jacobson, 1984), and *Edwardsiella tarda* (Wallace et al., 1966; White et al., 1973). However, the genus most commonly reported to cause disease in crocodilians is *Aeromonas* (for review see Marcus, 1981). This paper reports on the clinical signs of a disease in captive alligators in which blood or tissue cultures resulted in the isolation of several species of bacteria not usually reported as associated with infections in crocodilians.

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MATERIALS AND METHODS

All four alligators exhibiting clinical signs of disease (three males, one female; size range 1.5–2.2 m total length) were captive animals that were held between 2 wk and 15 mo (October 1983–September 1985) in a 15 × 20-m, flow-through, clay-lined experimental pond at the Savannah River Ecology Laboratory (SREL) in Aiken, South Carolina, USA. Water temperatures during this period ranged from approximately 0 to 30 C, and air temperatures from approximately −10 to 38 C. The maximum number of alligators in the pond at any one time was four. Diet during their confinement consisted mainly of chicken necks and backs, some beef heart and liver, and occasionally fish. All animals fed voraciously until the abrupt onset of illness. No signs of dietary deficiency were noted, either in animals that became ill or in others that had been maintained for up to 3 yr without showing clinical signs of disease.

Aseptic blood cultures were drawn via cardiac puncture in some of the animals that developed signs of disease and in four healthy individuals that were maintained also in captivity. Antibiotics were administered in the forelegs to avoid possible nephrotoxicity resulting from distribution directly to the kidneys from the hind limbs via the renal portal system. Dosages for antibiotics were administered at three 72-hr intervals as follows: tetracycline = 55 mg/kg body weight; amikacin sulfate = 7.8–9.3 mg/kg body weight; and gentamicin sulfate = 5–15.5 mg/kg body weight.

RESULTS

Clinical signs at the onset and throughout the course of the disease were consistent among all four study animals. For

each, sudden onset of anorexia was followed by lethargy and excessive basking behavior (prolonged exposure to ambient temperatures exceeding 35 C). Upon capture and isolation for closer examination, all showed some degree of damage to the integrity of the skin, resulting either from fights with other individuals, or, in one instance, from injuries received while crawling beneath a restraining fence. At initial examination, three of the four animals showed the pink coloration on the ventral surface that is suggestive of septicemia. Subsequent clinical signs in each animal depended on the course and timing of the treatment. In the three cases in which treatment (see following section for details) was not initiated earlier than 1 wk after initial signs became apparent, anorexia, lethargy, and excessive basking were followed by flaccid limb paralysis, stomatitis, dermatitis, and loosened teeth and claws. The fourth animal was treated immediately after anorexia became apparent, and subsequent signs noted in the other animals did not occur. Clinical signs for this animal subsided within 7 days after treatment was initiated.

The treatment regime for the first alligator (a 55-mg/kg i.m. injection of tetracycline at 3-day intervals for 1 wk) was based on the assumption that *Aeromonas* was responsible for the observed clinical signs. Although some improvement was initially noted, clinical signs recurred within less than 1 wk of termination of tetracycline injections, and the animal died 1 wk thereafter. Aseptic blood cultures had been drawn via cardiac puncture after recurrence of clinical signs, but test results were not returned until after the animal's death. These results showed the presence of *Citrobacter freundii* and *Enterobacter agglomerans* (family Enterobacteriaceae) both of which were found to be resistant to tetracycline. (A gross necropsy was attempted 6 mo after death, but results were inconclusive because of damage to tissues resulting from freezing.) Based on these results, the second animal to show the same clinical signs was treated at 3-day intervals with 15.5-mg/kg body weight i.m. injections of gentamicin sulfate, followed by a 7.8-mg/kg body weight, and 3 days later, a 9.3-mg/kg body weight i.m. injection of amikacin sulfate, both drugs being ones that tested effective against the aforementioned bacteria. (Amikacin sulfate was the drug of choice for the final injection because of its equal effectiveness and greater availability to us.) Stomatitis, already apparent, was treated topically with chlorhexidine and gentamicin sulfate. Clinical signs of disease began to subside within 3–5 days of initial treatment, and the animal resumed normal feeding and basking behavior 7–10 days thereafter. No blood cultures were performed on this animal. The third animal began to exhibit clinical signs within 2 wk of being placed in the experimental pond. At initiation of treatment, this animal already exhibited limb paralysis, stomatitis, and dermatitis. Aseptic blood samples were immediately drawn from the internal jugular vein, and 9.8-mg/kg i.m. injections of gentamicin sulfate were administered at 3-day intervals. However, no improvement was noted, and death ensued 6 days after signs were noted. Blood culture results, returned several days later, reported *Proteus* sp. and *Morganella morganii*. Sensitivity results showed that these bacteria were resistant to gentamicin sulfate.

Within 6 hr of this animal's death, a necropsy was conducted at the College of Veterinary Medicine, University of Georgia. Results indicated a widespread septicemia originating from skin wounds and subcutaneous abcesses that were apparently the result of fighting. Significant gross external lesions included multiple focal ulcers in the skin of the tail, body, and head. There were several subcutaneous abcesses: the largest (5-cm diameter), located at the point of the right shoul-

der and extending into the shoulder joint, was encapsulated and filled with greenish pus. Puncture wounds were associated with this abcess and with two others along the ventral midline. (No bacterial cultures had been attempted on any of the lesions.) Histological findings indicated these ulcers were filled with an exudate of fibrin and inflammatory cells that contained colonies of gram-negative bacterial rods. Internal findings included inflammatory lesions in the eye and lungs, swollen liver, ulcerated stomach, thrombosis related to disseminated intravascular coagulation, and intestinal hemorrhage, hepatic congestion, and grossly mottled kidneys, probably caused by shock. Pneumonia was present, and the air spaces in the lungs were filled with hemorrhage, edema fluid, and mixed inflammatory cells. Thrombi were present in the subcapsular sinusoids in the spleen and in the heart. The liver was congested, and congestion and necrosis were noted in the tips of the intestinal villi.

Based on these three cases, treatment of the fourth individual was begun immediately after anorexia and lethargy became apparent. Because the organisms responsible for this animal's illness were unknown, broad-spectrum gentamicin sulfate was still the drug of choice; a total of two 5-mg/kg i.m. injections were administered 3 days apart. Wounds were aseptically cultured, followed by disinfection with both chlorhexidine and povidone-iodine 10% antiseptic solutions. External wounds healed rapidly and the animal resumed a normal feeding and basking regime within 7 days. Culture results showed *C. freundii, Serratia marcescens*, and *Klebsiella oxytoca*; sensitivity results showed gentamicin sulfate to be the most effective antibiotic against these bacteria.

DISCUSSION

Most studies of reptilian diseases have not fulfilled Koch's postulates (Cooper, 1981); ours is no exception. Nonetheless, our results are significant because we have shown that bacteria other than *Aeromonas* can be associated with fatal infections in crocodilians. It is important to note that *Aeromonas* was never cultured from either blood or tissue samples taken from these four animals, even though the laboratory was specifically requested to test for this organism. Although we cannot positively determine whether these bacteria were primary pathogenic agents or secondary invaders, it seems likely that they are associated with the syndrome we have observed. None of the aforementioned bacteria were cultured from healthy alligators maintained either in our experimental pond or in natural environments. (Culture results from these apparently healthy individuals showed either negative bacterial growth or environmental contaminants, although *Aeromonas* was cultured from one individual that showed no signs of illness.) However, *C. freundii* also has been implicated in diseases of other captive crocodilians that showed clinical signs similar to those we noted (Brandt, pers. comm.; Collins, pers. comm.). Because cultures of the pond water taken in the autumn showed no significant bacterial growth, it seems likely that these bacteria were either present in the clay lining of the ponds, or that they were present in the pond water mainly during the warmer months of the year (May–September) when all infections occurred.

It is possible that the illnesses we observed may have been the result of an imbalanced diet. However, two lines of evidence suggest that dietary deficiencies were not involved in these cases. First, several animals have been maintained for more than 3 yr in these ponds on the same diet as that offered the affected animals, but showed no signs of illness. Second, two of the infected individuals developed signs of illness so soon after being placed in the ponds (<2 wk) that it seems unlikely that the available diet could have been respon-

sible for the disease. The common denominator for all four of the diseased animals was trauma (i.e., bites and abrasions) that damaged the integrity of the keratin layer covering the skin, and which may have allowed entry of the bacteria (Cooper, 1981). Further work is needed to determine the exact pathological progression of the disease.

Because our results suggested that infections in American alligators cannot always be attributed to *Aeromonas*, aseptic blood and wound cultures should always be taken on affected individuals. Puncture wounds and abcesses should be treated aggressively with antiseptic solutions to prevent subsequent infection. Our experience indicates that early detection and treatment with broad-spectrum antibiotics such as gentamicin sulfate or amikacin sulfate is preferable to waiting for identification and sensitivity results to be returned, particularly because reports have indicated that the reliability of sensitivity tests may be questionable (Cooper, 1981).

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LITERATURE CITED

ANONYMOUS. 1985. Service announces petition findings. U.S. Fish Wildl. Serv. Endang. Sp. Tech. Bull. 10(5): 8.

COOPER, J. E. 1981. Bacteria. *In* Diseases of the Reptilia, Vol. 1, J. E. Cooper and O. F. Jackson (eds.). Academic Press, London, England, pp. 165–191.

JACOBSON, E. R. 1984. Immobilization, blood sampling, necropsy techniques and diseases of crocodilians: A review. J. Zoo An. Med. 15: 38–45.

MARCUS, L. C. 1981. Veterinary Biology and Medicine of Captive Amphibians and Reptiles. Lea and Febiger, Philadelphia, Pennsylvania, 239 pp.

WALLACE, L. J., F. H. WHITE, AND H. L. GORE. 1966. Isolation of *Edwardsiella tarda* from a sea lion and two alligators. J. Am. Vet. Med. Assoc. 149: 881–883.

WHITE, F. H., C. F. SIMPSON, AND L. E. WILLIAMS, JR. 1973. Isolation of *Edwardsiella tarda* from aquatic animal species and surface waters in Florida. J. Wildl. Dis. 9: 204–208.